Remarks

Claims 1, 3, 10, 11 and 26-28 were pending in the subject application. By this amendment, Claim 1 has been amended, Claims 10 and 26 have been canceled without prejudice or disclaimer to applicant's right to pursue prosecution of these claims in a later-filed continuation or divisional application, and new Claims 29-33 have been added. The amendments to Claims 1 and the addition of new Claims 29-33 are supported by the claims as filed and the specification including, for example, at page 32, lines 14 and 14-19, and Examples 1-23. Accordingly, the amendments to Claim 1 and the addition of new Claims 29-33 do not introduce new matter, and their entry is respectfully requested.

In view of the preceding amendments and the remarks which follow, applicant respectfully requests that the Examiner reconsider and withdrawal the various rejections set forth in the Office Action, and solicits that the claims currently pending and under examination, namely Claims 1, 3, 11 and 27-33 are now in condition for allowance.

35 U.S.C. 102(b) Rejection

Claims 1, 3, 11, 27 and 28 were rejected under 35 U.S.C. 102(b) as anticipated by WO 01/64749 ("Kloetzer"). This rejection is respectfully traversed.

Kloetzer describes the use of an MIF antibody for treating arthritis, psoriasis, glomerulonephritis, septic shock, atopic dermatitis, and retinopathy associated with diabetes or lupus. Kloetzer does not teach a method of inhibiting the progression of type 1 diabetes in a mammal having type 1 diabetes, as set forth in Claims 1, 3, 11 and 27, or a method of inhibiting the development of type 1 diabetes in a mammal at risk for type 1 diabetes, as set forth in Claims 29-33. It is noted that a prophetic treatment of diabetic retinopathy is not a teaching that an MIF antibody would inhibit the progression of type 1 diabetes in a mammal having type 1 diabetes, or a teaching that an MIF antibody would inhibit the development of type 1 diabetes in a mammal at risk for type 1 diabetes. It does not necessary follow that a proposed treatment of diabetic retinopathy

would necessarily result in inhibiting the progression of type 1 diabetes in a mammal having type 1 diabetes, or in inhibiting the development of type 1 diabetes in a mammal at risk for type 1 diabetes. Accordingly, Kloetzer does not anticipate the claimed invention, and reconsideration and withdrawal of this rejection is respectfully requested.

35 U.S.C. 103 Rejections

1. Claims 1, 3, 9, 11 and 27 were rejected under 35 U.S.C. 103 as being unpatentable over Bojunga, et al. in view of Nishirira. This rejection is respectfully traversed.

Bojunga suggests that MIF may play a possible role in autoimmune-inflammatory events such as type-1 diabetes. In this regard, this potential role was based on preliminary studies in Bojunga in which it was shown that MIF-mRNA expression was elevated in the splenic lymphocytes of NOD mice in which diabetes was spontaneously induced. However, the MIF protein levels in the diabetic animals were less than in the normal controls. In another set of experiments, Bojunga evaluated the effect of MIF-protein treatment on diabetes in the NOD mice. While MIF treatment led to an increase in diabetes incidence over the untreated animals, Bojunga stated that this trend was not statistically significant. In summary, Bojunga suggests that MIF may be involved in diabetes. However, Bojunga does not provide any data which supports that an agent that inhibits a macrophage migration inhibitory factor (MIF) in the mammal, wherein the agent comprises a binding site of an antibody that binds specifically to MIF, would be effective in inhibiting the progression of type 1 diabetes in a mammal having type 1 diabetes or in inhibiting the development of type 1 diabetes in a mammal at risk for type 1 diabetes.

The addition of Nishirira does not remedy the deficiencies of Bojunga. Nishirira describes MIF as a target molecule in multiple sclerosis. Nishirira does not teach or suggest that an agent that inhibits a macrophage migration inhibitory factor (MIF) in the mammal, wherein the agent comprises a binding site of an antibody that binds specifically to MIF, would be effective in inhibiting the progression of type 1 diabetes in

a mammal having type 1 diabetes or in inhibiting the development of type 1 diabetes in a mammal at risk for type 1 diabetes.

For these reasons, the claimed invention is patentable over Bojunga in view of Nishirira. Accordingly, reconsideration and withdrawal of this rejection is respectfully requested.

2. Claim 28 was rejected under 35 U.S.C. 103 as being unpatentable over Bojunga, et al. in view of Nishirira as applied to Claims 1, 3, 11 and 27 above, and further in view of U.S. Patent No. 5,530,101 ("Queen"). This rejection is respectfully traversed.

As discussed above, the claimed invention is patentable over Bojunda in view of Nishirira. The addition of Queen does not remedy the deficiencies of Bojunda and Nishirira. Queen describes the production of humanized antibodies. Queen does not teach or that an agent that inhibits a macrophage migration inhibitory factor (MIF) in the mammal, wherein the agent comprises a binding site of an antibody that binds specifically to MIF, would be effective in inhibiting the progression of type 1 diabetes in a mammal having type 1 diabetes or the development of type 1 diabetes in a mammal at risk for type 1 diabetes.

For these reasons, the claimed invention is patentable over Bojunga, Nishirira and Queen. Accordingly, reconsideration and withdrawal of this rejection is respectfully requested.

Information Disclosure Statement

In form PTO/SB/08b attached to the Office Action, certain citations were lined through for missing the authors' names and publication dates. The undersigned does not understand the relevance of requiring authors' names and publication dates for the Search Reports and IPER listed on form PTO/SB/08b, since the authors' names and publication dates do not seem relevant to patentability. In addition, the August 29, 2008 Notice of Acceptance, which indicated receipt of the International Search Report and the

IPER, didn't identify these documents by authors or publication dates. The mailing dates of the Search Reports and IPER and their originating application nos. (for the corresponding European application or the underlying PCT application) are indicated on form PTO/SB/08b, and it is believed that this should be sufficient for their identification.

No fee, other than the $555 three month extension of time fee and the $405 RCE fee, is deemed necessary in connection with the filing of this Amendment. If this fee is in any way deficient, or if any additional fee is required to preserve the pendency of the subject application, authorization is hereby given to charge any such fee to Deposit Account No. 01-1785.

Respectfully submitted,

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